

## ***Expert Commentary Series***

# **Wrong About Genetic Research and Autism: A Review of Lyn Redwood's Article "Science as a Means of Social Control" (*SafeMinds*, August 23, 2013)"**

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### **Abstract**

A number of organizations as well as bloggers have arisen over the past several decades claiming that vaccines and/or their ingredients cause a number of disorders. The results of their efforts have been a decline in vaccine coverage and a rise in previously rare childhood diseases, resulting in unnecessary suffering, hospitalizations, long term disabilities, and even death. The following paper will demonstrate, using one article by Lyn Redwood, co-founder of *SafeMinds* and a leading figure among anti-vaccinationists, the poor scholarship and science displayed by many antivaccinationists. If people are to decide on whether to vaccinate their children or not, it should be based on scholarly, well-grounded science, and reflect basic common sense, not claims made by people who are deficient in these.

A recent article/post by Redwood on *SafeMinds*, "Science as a Means of Social Control," should raise a number of red flags regarding her scholarship, basic understanding of science and common sense and, given her key position in *SafeMinds*, of the overall credibility of the organization. Redwood's article claims that a recent study in the journal *Science* by Rietveld *et al.*, "GWAS of 126,559 Individuals Identifies Genetic Variants Associated with Educational Attainment," found that environment contributed 98% to educational attainment and genetics only 2% and, by implication, the same applied to Autism Spectrum Disorders.

The conclusions of this paper are:

1. Redwood found an article on the online magazine *Truthout* by Jonathan Latham, "Science as Social Control: Political Paralysis and the Genetics Agenda," that discussed the Rietveld *et al* study, and based her own article on Latham's. However, a careful reading of Rietveld *et al*'s article suggests that either Redwood did not bother to read the easily available Rietveld *et al*'s article and supplement or did not understand them. The Latham article that she based her article on was wrong about the Rietveld *et al*'s study findings.
2. Redwood's claim that 98% of educational attainment is accounted for by environmental factors is not what the Rietveld *et al* study found which attributed 40% to genetics, thus 60% to environment. Redwood's claim is so extreme that one would expect a scholarly scientific rendition of peer-reviewed findings, not "what [she] instinctively knew."
3. Redwood's article contradicts an earlier article she herself co-authored, "Autism: A Novel Form of Mercury Poisoning," where genetics was considered a significant component of autism.
4. Redwood rejects the progress being made on the genetic contribution to various aspects of human personality, cognitive abilities, and behaviors, including Autism Spectrum Disorders (ASD), that has already led to a variety of interventions and the prospect of many more to come. In addition, she ignores the evidence that half of our genome is mainly devoted to our brains.
5. Redwood seems to assume that the genetic findings for educational attainment would apply to Autism Spectrum Disorders, ignoring the extensive research evidence that genetic vs environmental contributions and their interactions vary widely between traits, cognitive abilities, and behaviors.
6. Redwood misunderstands that *de novo* mutations, while not present in either parent, are mutations in the germ line, sperm and ova, prior to conception. These mutations

often can be random or associated with environmental factors, e.g. toxins, infections, radiation.

7. Redwood would like us to believe that finding genetic contributions will lead to “blaming the victim,” always possible among some; but this is not a prevailing attitude and certainly NOT the goal of researchers. Researchers’ goals, rather, are to improve diagnosis and both prevention and treatment.

As stated on their website: “SafeMinds ultimate goal is to find the truth - - to encourage and support efforts to conduct medical research that provide credible findings to support that the mercury-autism hypothesis is true.” They obviously don’t see the contradiction between having “the ultimate goal . . . to find the truth [and] to support that the mercury-autism hypothesis is true.” In my opinion, Redwood’s mind is made up. She is absolutely certain she is right so she searches the internet to find something that supports her position, even if just one paper, without thoroughly vetting it.

Her article shows the desperate lengths that she will go to. Redwood does not apply a scholarly scientific approach and even ignores common sense. Why would anyone accord what she writes any credibility? And if, as a co-founder of *SafeMinds* as well as a driving force and advocate for their position, she represents their standard of writing on scientific matters, how can anyone accord anything *SafeMinds* writes any credibility?

## Introduction

A number of organizations as well as bloggers have arisen over the past several decades claiming that vaccines and/or their ingredients cause a number of disorders. Foremost among these is autism. The results of their efforts have been a decline in vaccine coverage and a rise in previously rare childhood diseases, resulting in unnecessary suffering, hospitalizations, long term disabilities, and even death. If one is to believe their claims regarding vaccines, the first question that comes to mind is whether the people who make these claims employ acceptable standards of scholarship, science, and, in some cases, even basic common sense. In other words, do they know what they are talking about?

*SafeMinds*, co-founded by Lyn Redwood, is one of the more active and vocal of the antivaccinationist organizations. The following paper will show, using one article posted on *SafeMinds* website as an example, the poor scholarship and science displayed by many antivaccinationists. If one is to presume that, in her role as a co-founder of and staunch advocate for *SafeMinds*, she represents the organization's standard of writing on scientific matters, how can anyone accord anything the organization writes any credibility? If people are to decide on whether to vaccinate their children or not, it should be based on scholarly, well-grounded science, and reflect basic common sense, not claims made by people who are deficient in these.

## Background

According to *SafeMinds*:

"Safeminds was founded to raise awareness, support research, change policy and focus national attention on the growing evidence of a link between mercury and neurological disorders. . . In April of 2000, SafeMinds founders put forth the first definitive work reviewing the link between mercury and Autism Spectrum Disorders." (SafeMinds, "About SafeMinds") (<http://www.safeminds.org/about-2/>)

"SafeMinds . . . Established the link between mercury and autism through the landmark paper, 'Autism, A Novel Form of Mercury Poisoning.' SafeMinds is the driving force pushing forward science that links environmental factors, such as mercury, to autism. . . SafeMinds ultimate goal is to find the truth -- to encourage and support efforts to conduct medical research that provides credible findings to support that the mercury-autism hypothesis is true." (Safeminds "Accomplishments") (<http://www.safeminds.org/about-2/accomplishments/>)

Lyn Redwood, R.N., M.S.N., is co-founder and board member of the Coalition for SafeMinds and the National Autism Association and a co-author on the paper that launched *SafeMinds*, "Autism, A Novel Form of Mercury Poisoning."

## **Lyn Redwood's "Science as a Means of Social Control" (August 23, 2013)**

A recent article/post by Redwood should raise a number of red flags regarding her scholarship, basic understanding of science and common sense and, given her key position in *SafeMinds*, of the overall credibility of the organization.

Redwood writes:

For years now I have been trying to understand why the huge focus and investment on genetics in autism research when to date the findings have been dismal. I also could not understand why promising research investigating environmental factors have been dismissed or worse yet, conducted in a manner to purposefully manipulate significant finding away from their connection to certain risk factors (like mercury or vaccines). Then I came across this article, <http://truth-out.org/news/item/17916-science-as-social-controlpolitical-paralysis-and-the-genetics-agenda> which explained perfectly what I instinctively knew, science is being used as a means of exonerating industry and/or government for culpability by blaming the individual for having the poor luck of bad genetics.

The author of the article reviews a recent publication in the prestigious journal *Science* which investigated whether variations in individual "educational attainment" (essentially, whether students complete high school or college) could be attributed to inherited genetic differences. According to the research, fully 98% of all variation in educational attainment is accounted for by factors other than a person's simple genetic makeup. But nowhere in the title of the paper, the accompanying press release or in the summary was this important fact even mentioned! The focus was on the identification of three gene variants that each contributed 0.02% to the variation in educational attainment.

The high prevalence of de novo findings (new genetic abnormalities not found in the mother or father) supports the logical conclusion that there is a large environmental component to autism

spectrum disorders. And more importantly, identifying environmental triggers that lead to autism is the only strategy for mitigating harm and preventing autism. (Redwood, 2013)

Over the years I have read in numerous papers and books that current and historical research has found the genetic component of intelligence and related cognitive abilities to be around 50 percent (e.g. Lewis, 2005, p.140, Rietveld, 2013a, p.4). In addition, “more than half the genome is put to work primarily or exclusively in the brain.” (Pinker, 2012, p. 91) Since current estimates of the total number of genes in the human genome hover around 20,000 - 25,000 (National Human Genome Research Institute, 2001), that means about 10,000 - 12,500 genes are mainly devoted to development of the human brain. It therefore seems highly likely that more than 3 Single Nucleotide Polymorphisms (SNPs, see Technical Section) would contribute to educational attainment. However, I am always open to new research. That doesn’t mean that one or two publications will automatically change my mind; but if they are well-done methodologically, subject to peer-review, and replicated, I have absolutely NO problem rejecting previous scientific findings, regardless of how long I have held them. So, I decided to further research Redwood’s claim that “fully 98% of all variation in educational attainment is accounted for by factors other than a person’s simple genetic makeup,” especially given the extremity of this claim.

While Redwood’s rendition of the article by Jonathan Latham in the online magazine *Truthout* (Latham, 2013) is accurate, why did she rely on a magazine article when both the original article and supplementary materials were available online to be downloaded free in PDF format? (Rietveld, 2013ab) In addition, the journal *Science* is available in almost all university, community college, and public libraries and through online electronic databases such as *ScienceDirect* available from home for patrons with library cards.

### **So what does the Rietveld *et al* article actually say?**

“Estimates suggest that around 40% of the variance in educational attainment is explained by genetic factors (5). Furthermore, educational attainment is moderately correlated with other heritable characteristics, including cognitive function and personality traits related to persistence and self-discipline.” (Rietveld, 2013a, p. 1467) The “(5)” refers to a footnote that

states “See the supplementary materials on *Science* Online.” In this same paper in *Science*, the authors state: “A linear polygenic score from all measured SNPs accounts for  $\approx 2\%$  of the variance in both educational attainment and cognitive function.” This 2% figure is the likely source of Latham’s statement that “98% of all variation in educational attainment is accounted for by factors other than a person’s simple genetic makeup,” a statement that Redwood repeats in her article. However, anyone reading the original *Science* paper and supplement, even those lacking training in genetics, would likely see the disconnect between the authors’ citation of 40% and 2% to describe the degree to which genetic factors contribute to variance in educational attainment (for those interested see Technical Section below). Why Jonathan Latham in his online magazine article, and Redwood in her parroting of that article, seized on the 2% figure and ignored the 40% figure is without explanation.

Furthermore, Redwood’s insistence on the 2% figure is also at odds with her own article that launched *SafeMinds* which stated: “studies in mice as well as humans indicate that susceptibility to Hg effects arises from genetic status, in some cases including a propensity to autoimmune disorders. ASD [Autism Spectrum Disorders] exhibits a strong genetic component, with high concordance in monozygotic twins and a higher than expected incidence among siblings; autism is also more prevalent in families with autoimmune disorders.” (Bernard, 2001, pp. 466-7) Nowhere does she address this change in her position.

In addition, human traits, cognitive abilities, and behaviors range in the amount of variance attributed to genes. Research certainly reflects this (e.g. Lewis, 2005, pp. 140-141). Redwood apparently assumes that research findings of the heritability of educational attainment should automatically apply to autism. Keep in mind that the Rietveld paper did not include severely developmentally challenged individuals whose intellectual abilities reflect both quantitative and qualitative differences that potentially reflect genetic differences.

## **Are the Results of Research on Genetics and Autism “Dismal” and Should We Invest Solely in Research on Environmental Triggers?**

Redwood writes: “For years now I have been trying to understand why the huge focus and investment on genetics in autism research when to date the findings have been dismal. . . and more importantly, identifying environmental triggers that lead to autism is the only strategy for mitigating harm and preventing autism.” (Redwood, 2013). First, while genetic research is making progress, it is still in the early stages. As explained below, Redwood’s assessment of the research on genetics and autism has no basis in reality. Using Redwood’s approach to scientific research, that is, a short term perspective, much of what we have learned about numerous disorders and genetics would have likely never been achieved.

This is not the place to give a thorough review of genetics and autism; however, genetics research is essential to both developing prevention strategies and treatments. As the following shows, there have been major advances in assessing the genetics of autism and using these advances to develop, among other things, targeted treatments. The abstract from a recent article states: “There have been recent advances in the understanding of the underlying pathophysiology of ASD pertaining to genetics, epigenetics, neurological, hormonal, and environmental factors that contribute to the difficulties found in individuals with ASD. With this improved understanding, there has been a shift in the application of psychopharmacology in ASD and its related disorders.” (Sung, 2014)

Another recent article states: “Strong evidence for genetic causes of autism implicates proteins that mediate synaptic transmission and structure. . . Promising pharmacological targets . . . are now being pursued in early clinical trials. . . Synaptic genes predict pharmacological targets for therapeutic interventions in autism.” (Silverman, 2013, pp. 1-2) The article includes a list of current drugs being investigated.

And another recent review states: “Autism spectrum disorder (ASD) represents a heterogeneous group of neurodevelopment disorders . . . leading some researchers to refer to these various disorders as ‘the autisms’. . . Sequencing of both common polymorphisms, which have a small effect on ASD susceptibility, and rare genetic variation, which has a larger effect on



the development of ASD.” (Jeste, pp. 74-75) “The ACMG [American College of Medical Genetics] estimates that the total diagnostic yield (percentage of children in whom a test will yield positive, clinically relevant information) of performing the above recommended genetic testing in children with ASD is 40% . . . After a diagnosis of ASD has been made, the primary goal from a clinical standpoint is to maximize a child’s potential for cognitive and functional gains. The rapid advances in genetics have facilitated an understanding of developmental trajectories, comorbidities and biological mechanisms underlying the deficits in ASD which, in turn, will open the door to the development of more mechanism-based, phenotype-specific treatments for these children.” (ibid, p. 79) (Note. see also Technical Section “**Heritability vs. Specific Genes Used as Covariates**)

Redwood also claims that: “The high prevalence of *de novo* findings (new genetic abnormalities not found in the mother or father) supports the logical conclusion that there is a large environmental component to autism spectrum disorders.” While no one denies the role of environmental factors, Redwood also misinterprets the meaning of “*de novo*.” According to a recent review, “since 2007, studies have shown a strong source of causality for ASDs, namely *de novo* mutations (that is, new mutations) that originate in the parental germ line.” (Ronemus, 2014, p. 133). And “an open question is whether these mutations occur mainly in the germline, during embryogenesis or somatically. A number of studies have shown an apparent germline origin of mutations.” (Veltman, 2012, p.565) In other words, the *de novo* mutations may not be “abnormalities . . . found in the mother or father”; but in their germ lines, i.e. ova or sperm, with causes including random mutations. Whether future research will confirm this or not, it is certainly premature to dismiss it.

Genes can be expressed or silent. They can experience mutations, some with no effect, some negative, and some positive. Genes interact with each other and with the environment. Genes vs environment should not be seen as a dichotomy, research needs to be directed at both. Many disorders with a strong genetic component involve an environmental component, though in the case of ASD, the scientific evidence is that if there is such a component, its role is prenatal. As advances are made in gene sequencing, we will discover more and more genes involved in complex disorders, including those subsumed under the heading Autism Spectrum Disorders. Some genes will be subject to gene therapies, some will be targets for specific medicines that “turn them on or off,” or modify their protein products, and

some will give clear evidence for the risks of various environmental chemicals and toxins in subgroups of the population. We are only on the cusp of potential genetic and epigenetic advances and Redwood's flippant dismissal of genetic research is unscientific, to say the least.

As for Redwood's claim that genetic research will lead to "blaming the individual for having the poor luck of bad genetics," this is preposterous. Do you know of anyone who blames people with genetic disorders for their condition, or blames the parents in any sense of attributing culpability to them?

In Steven Pinker's fascinating book, *The Blank Slate: The Modern Denial of Human Nature* (2002), he writes:

When it comes to explaining human thought and behavior, the possibility that heredity plays any role at all still has the power to shock. To acknowledge human nature, many think, is to endorse racism, sexism, war, greed, genocide, nihilism, reactionary politics, and neglect of children and the disadvantaged. Any claim that the mind has an innate organization strikes people not as a hypothesis that might be incorrect but as a thought it is immoral to think. . . . In some cases, an extreme environmentalist explanation is correct: which language you speak is an obvious example . . . . In other cases, such as certain inherited neurological disorders, an extremely hereditarian explanation is correct. In most cases the correct explanation will invoke a complex interaction between heredity and environment. . . . Acknowledging human nature . . . does not . . . require one . . . to accept current levels of inequality. . . . The refusal to acknowledge human nature . . . distorts our science and scholarship, our public discourse, and our day-to-day lives. . . . The dogma that human nature does not exist, in the face of evidence from science and common sense that it does, is just such a corrupting influence. (Pinker, 2002, pp. viii-ix)

Personally I find Redwood's statement of "blaming the victim" offensive.

## To summarize:

1. Redwood found an article on the online magazine *Truthout* by Jonathan Latham, "Science as Social Control: Political Paralysis and the Genetics Agenda," that discussed the Rietveld *et al* study, and based her own article on Latham's. However, a careful reading of Rietveld *et al*'s article suggests that either Redwood did not bother to read the easily available Rietveld *et al*'s article and supplement or did not understand them. The Latham article that she based her article on was wrong about the Rietveld *et al*'s study findings.
2. Redwood's claim that 98% of educational attainment is accounted for by environmental factors is not what the Rietveld *et al* study found which attributed 40% to genetics, thus 60% to environment. Redwood's claim is so extreme that one would expect a scholarly scientific rendition of peer-reviewed findings, not "what [she] instinctively knew."
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often can be random or associated with environmental factors, e.g. toxins, infections, radiation.

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As stated on their website: “SafeMinds ultimate goal is to find the truth - - to encourage and support efforts to conduct medical research that provide credible findings to support that the mercury-autism hypothesis is true.” They obviously don’t see the contradiction between having “the ultimate goal . . . to find the truth [and] to support that the mercury-autism hypothesis is true.” Put another way, their goal is to prove a hypothesis, not test a hypothesis. Someone flunked Science 101. In my opinion, Redwood’s mind is made up. She is absolutely certain she is right so she searches the internet to find something that supports her position, even if just one paper, without thoroughly vetting it.

Her article shows the desperate lengths that she will go to. Redwood does not apply a scholarly scientific approach and even ignores common sense. Why would anyone accord what she writes any credibility? And if, as one of the founders of *SafeMinds* as well as a driving force and advocate for their position, she represents their standard of writing on scientific matters, how can anyone accord anything *SafeMinds* writes any credibility?

## Technical Section

With regard to the discrepancy between the 40% and 2% figures to describe the degree to which genetic factors contribute to variance in educational attainment, one needs to actually read the original paper in *Science* (“GWAS of 126,559 Individuals Identifies Genetic Variants Associated with Educational Attainment”)

(<http://www.sciencemag.org/content/340/6139/1467.full.pdf>), which is the basis for Latham’s magazine article, which in turn was the basis for Redwood’s post “Science as a Means of Social Control” (August 23, 2013).

In Section 2 of the Supplementary Materials “The heritability of educational attainment,” (pp. 7-10) the authors state their use of “the Multi-Generation Registry of all Swedish males born between 1950 and 1969, as well as their full brothers and half-brothers (regardless of birth year)” together with Statistics Sweden’s [Swedish National Agency] administrative records to measure years of education and the National Service Administration that contains a battery of mental tests that, at the time, all Swedish men were required by law to take as part of their military conscription. Based on three different models, the heritability estimates [ $h^2$  represents the percentage attributed to heritability] for educational attainment were  $h^2 = 0.552$  (s.e. 0.027,  $N = 216,091$ ),  $h^2 = 0.494$  (s.e. 0.045,  $N = 207,738$ ), and  $h^2 = 0.556$  (s.e. = 0.030,  $N = 207,738$ ). In addition, Table S10 in the Supplementary Materials lists “Previously published twin study findings on the heritability of educational attainment.” (p. 111) The “around 40% of the variance in educational attainment is explained by genetic factors,” represents a conservative estimate based on the current study’s findings and 11 previous studies listed in Table S10.

So where does the 2% and 0.02% for single gene variations come from and what do these numbers mean? To understand this, one has to understand the various approaches to genetic analysis:

Genetic variance for a polygenic trait is mostly due to the additive effects of recessive alleles of different genes. For some traits, a few dominant alleles can greatly influence phenotype, but because they are rare, they do not contribute greatly to heritability. . . Epistasis (interaction between alleles of different genes) can also influence heritability. Geneticists calculate a ‘narrow’ heritability that considers only additive recessive effects, and a ‘broad’ heritability that also considers the effects of rare dominant alleles and epistasis. . . Two special types of people . . . can help geneticists to tease apart the genetic and environment components of multifactorial traits—adopted individuals and twins.(Lewis, 2005, p.141)

With the availability of more types of genetic markers and genome sequence data, researcher have more defined tools to identify DNA sequences. . . SNP, or single nucleotide polymorphisms, is a site within a DNA sequence that varies in at least 1 percent of a population. . . SNPs are useful in **association**

**studies**, in which researchers compare SNP patterns between a group of individuals who have a particular disorder and a group who do not.’ (Lewis, 2005, p. 143)

[Just to be clear] Narrow heritability is the fraction of variance that can be accounted for in aggregate by the cumulative additive effects of all genetic polymorphisms. . . its best linear genetic predictor; that is, a predictor in which each polymorphism enters additively, and the effect of each polymorphism is constrained to be linear in the number of reference alleles. Broad heritability, which is necessarily larger, is the fraction of variance . . . that can be explained in aggregate by all genetic factors . . . best genetic predictor, allowing not only for linear and additive effects but also for interactions among different polymorphisms (‘epistasis’) and nonlinear effects of specific polymorphisms (‘dominance’). . . Lykken proposed that for SWB (along with several other traits including personality), most—if not all—of the genetic influences stem from higher-order epistatic interactions among genetic polymorphisms. Individual polymorphisms . . . that are rare in the population—which may collectively contribute much of the narrow heritability—will be much more difficult to reliably detect than polymorphisms that are common in the population. . . [An even narrower analysis] that cannot be estimated from twin or family data and that is necessarily smaller than narrow heritability, namely common narrow heritability: the fraction of variance that can be accounted for in aggregate by the cumulative additive effects of genetic polymorphisms that are common in the population (typically defined as minor allele frequency > 1%). (Rietveld, 2013a, p. 2)

So, what did the authors actually do? “After quality control, a total of 2,515,021 autosomal SNPs were meta-analyzed across 72 input files for *EduYears*. For *College* 2,510,021 autosomal SNPs were meta-analyzed across 65 input files. Only SNPs with an availability of  $\geq 80\%$  in the total sample were selected, resulting in 2,299,174 SNPs for *EduYears* and 2,309,290 SNPs for *College*. . . SNPs with  $p$ -values  $< 10^{-6}$  in the discovery stage were brought forward for further analysis in the replication stage.” (Rietveld, 2013b, p. 6) Thus, the 3 genome-wide significant SNPs are an extremely small subset of the subset that was forwarded to the replication stage. Because they meta-analyzed GWAS results from 54 samples in to-

tal, they did not look into specific results within one sample; but to the overall result across all samples. This was necessary because only with a large enough sample did they have sufficient power to detect small effects.

The 0.02% represented individual SNPs and the 2% is from all measured SNPs (roughly 2 million). Keep in mind that the human genome consists of approximately 3 billion DNA base pairs. Each pair represents two nucleotides, thus 6 billion nucleotides altogether. In other words, the roughly 2 million SNPs measured by the authors represents a very small percentage of all possible SNPs and, in addition, they analyzed them linearly, not taking into account any interactions. The authors go on to state: “The seven loci that did not reach genome-wide significance did not replicate (the effect went in the anticipated direction in five out of seven cases).” (Rietveld, 2013b, p 1467). Thus, “if the size of the training sample used to estimate the linear polygenic score increased, the explanatory power of the score in the prediction sample would be larger . . . An asymptotic upper bound for the explanatory power of a linear polygenic score is the additive genetic variance across individuals captured by current SNP microarrays. Using combined data . . . we estimate that this upper bound [the common narrow heritability] is 22.4% (SE = 4.25) in these samples.” (Rietveld, 2013b, p. 1469) [Note. Derived from Table S12 in the Supplementary Materials] Their sample size (despite being over 100,000) just wasn’t big enough to estimate the contribution of most of the SNPs very precisely.

**Heritability vs. Specific Genes Used as Covariates** (the following based on Manski, 2011):

Heritability is based on the equation,  $y = g + e$ , where  $y$  is the predicted trait or cognitive ability,  $g$  is the genetic component, and  $e$  is the environmental component. Heritability, based on twin studies and other similar types of studies, has sometimes been used to determine which is “more important” in order to determine social policy. “The ratio of the population variance of  $g$  to the variance of  $y$  is called the heritability of  $y$  . . . The equation specifies a production function in which  $g$  and  $e$  contribute additively to outcomes, rather than interact with one another” (Manski, 2011, pp. 84-85)

Technological progress in gene measurement has increasingly enabled collection of data on the expression of specific genes in large samples of individuals. With the advent of gene sequencing, making treatment choice conditional on observed co-

variates is referred to as *targeting* or *profiling*. . . Gene measurements may be informative about treatment response. If it were found that the outcomes of medical treatments or educational interventions vary systematically across persons with different observed genes, then physicians or school counselors may want to condition treatment decisions on these covariates. . . Econometricians and statisticians have long sought to prescribe effective approaches to conditional prediction and analysis of treatment response when the number of observed covariates is large relative to the size of the available sample of persons. Perhaps the simplest and most common practice is to a priori choose a reasonably small subset of the observed covariates and use this subset as the conditioning variables, ignoring the other covariates. This practice is legitimate. (Manski, 2011, pp 89-91)

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